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The optics of noncommunicable diseases: From lifestyle to environmental toxicity

Introduction

In September 2018, the United Nations held its third High Level Meeting on noncommunicable diseases (NCDs). As a think piece circulated by the World Health Organisation (WHO) in advance of the meeting warned, ‘the world is now reaching an inflection point’ in its efforts to achieve its Sustainable Development Goals on health (WHO, 2018a, 4). This was especially acute because despite widespread cries of global “crisis” from the public health community (Marrero et al., 2012, Beaglehole et al., 2011), progress on tackling NCDs has long been stymied by a lack of funding, political commitment and accountability mechanisms (Horton, 2005, Blundell and Hine, 2018, Adjaye-Gbewonyo and Vaughan 2019). Recently, criticism has sharpened with the nomenclature and framing of NCDs coming under sustained fire for propagating the ‘misnomer’ that their causes are ‘individual rather than societal’ and for placing an ‘unhelpful emphasis on individual healthy choices’ rather than the wholesale reconfiguration of ‘social and policy environments’ (Allen and Feigl, 2017a). In other words, and as I will explore in this paper, the *optics* of the ‘NCD crisis’ are wrong. While the Political Declaration agreed in October 2018 may have been critiqued for its continued adherence to causal narratives of behavioural risk (Hunter et al., 2010), it also marked a profound break that begs further exploration. For the first time since the NCD category was simplistically constructed as four diseases and four shared (modifiable, behavioural) risk factors, mental health was added to the disease outcomes and air pollution to the risk factors. The “4 x 4” shorthand became the “5 x 5” (Horton, 2018; Linou et al, 2018) and, in the process, transformed the NCD category into a fundamentally *different form* of ‘information infrastructure’ (Bowker and Star, 2000, 47) that now communicates itself in different ways across ‘disparate communities’ (2000, 286).

The inclusion of air pollution as an explicit risk factor is itself an important inflection point as it forcibly elides two diverse aetiological paradigms: *behavioural* and *environmental* readings of risk and disease causality. This forces previously siloed research traditions – public health and environmental health - into far closer contact. It also, I argue, shifts the locus of health risk from *acts of consumption* to *conditions of exposure*. In so doing, it complicates political narratives of individual responsibility for health that have long been applied to the context of NCDs and instead raises questions concerning the inequities and spatial distribution of ‘the white noise of low-level exposure’ (Hepler-Smith, 2019, 450; see also Cordner, 2016). Although the built and natural environment has long been an implicit segment of the NCD agenda (i.e. obesogenic environments), it has never been conceived as an explicit and named *risk factor* for the *category*. Yet, it remains ‘notoriously hard to demonstrate environmental causation of illness, given the limitation of pre-existing research bases, not to mention the multiplicity of possible causal agents’ (Buell, 1998, 660). Indeed, the ‘elusive, ambiguous relationship between chronic illness and long-term exposures to environmental pollution’ (Markowitz and Rosner, 2013, 289) is further complicated by the newly expanded array of ‘commercial determinants of health’ (Kickbusch et al., 2016) that seek to evade the kinds of regulation that would protect the public (Oreskes and Conway, 2010). In critically reflecting on the conceptual implications of the re-categorisation of NCDs, this paper opens new interdisciplinary research agendas and points of theoretical connection.

This is important as research traditions on the causal pathways of the diseases that compose the NCD category are deeply siloed. For example, the long history of environmental health research linking toxic exposures with chronic disease outcomes has been largely absent in the NCD agenda. By contrast, public health research has overwhelmingly couched health risk in terms of ‘unhealthy’ behaviours and their upstream structural or ‘social determinants’ (Marmot, 2005). When the ‘environment’ emerges in this research tradition, it tends to

represent a shorthand for the regulatory backdrop, socio-economic and political conditions of possibility that shape consumption practices, inequities or contexts of health risk (Hill and Peters, 1998). This contrasts sharply with environmental health's ecological consideration of the combined built and natural environments and their influence on human biological functioning. In the former vision, *consumption* (of risky products and lifestyles) is the main risk to health. In the latter, *exposure* to the contaminated environment itself is the source of risk through the 'chemical cocktail of industrial pollutants in the air we breathe, the food we eat, the water we drink and the products we handle everyday' (Cordner, 2016, 5). These aetiological (and research) traditions have rarely combined in the global NCD agenda, despite both sharing a concern with the origins and genesis of the same sets of chronic diseases.

Given this, there is scope to reflect on how we might now usefully consider NCDs through an *optics of toxicity* rather than previous framings that have perniciously and ineffectually coalesced around individual responsibility, lifestyle and choice. Although the term 'optics' has become ubiquitous in the lexicon of politics and business – used to connote public understanding or perception of an event and the political consequences of this - the term rarely appears in relation to health despite its obvious conceptual utility (although see Benton, 2017, Gostin, 2014 on Ebola). And, as the numerous, interdisciplinary critiques of the failure to address NCDs makes clear, there may be something fundamentally lacking in the optics of the category that ensures continued political inaction (Allen and Feigl, 2017b, Sridhar et al., 2011). Toxicity, in turn, complicates the ascription of individual agency in relation to NCDs, forces us re-think the 'lifestyle' paradigm and calls into question the temporality and spatiality of cause-effect in relation to human health. To explore these issues, I first analyse the circuitous policy manoeuvres that have led to the inclusion of air pollution as a risk factor for NCDs before turning to how NCDs might be usefully (re)considered through the lens of toxicity. I then examine the consequences of this for public health opposition to the 'commercial

determinants of health’ (Kickbusch et al., 2016) and how this might feed into a new optics of NCDs focussed on *exposure* rather than *consumption*, before offering up some conclusions.

The “4 x 4” becomes the “5 x 5”

For the past decade, the NCD category has been made up of four diseases (cancer, cardiovascular disease, diabetes and chronic respiratory disease) and the four shared, “modifiable” behavioural risk factors of smoking, diet, physical activity and alcohol. It is worth noting that when the first *Global Strategy* on NCDs was published by the WHO in 2000, alcohol was not included as a behavioural risk factor, however by the 2008-2013 *Global Action Plan*, alcohol was listed as the fourth risk factor and the implausibly symmetrical 4 x 4 shorthand was born. In 2017, the WHO’s Director-General convened an Independent High-Level Commission on NCDs. Public health hopes for the final report were high, especially given the backdrop of the two UN High Level Meetings on NCDs in 2011 and 2014, a planned third in Autumn 2018 and the publication of the *Montevideo Roadmap 2018-2030 on NCDs as a Sustainable Development Priority*. Yet, when the final report – *Time to Deliver* (WHO, 2018c) - was released one month later, criticism from stakeholders and the public health community was swift.

By the time of *Time to Deliver*’s release, environmental health researchers and epidemiologists had been demonstrating links between pollution and human health for decades, including those diseases that comprise the NCD category such as cardiovascular disease and cancer (see for example Ezzati, 2005, Davis, 2002). Give this, it was unsurprising that one branch of critique was *Time to Deliver*’s lack of any explicit recommendations with respect to the environmental drivers of NCDs (Chowdhury et al., 2018). Instead, it states only that ‘there is *increasing evidence* about the role of indoor and outdoor air pollution, with its links to urbanization, in the

development of NCDs’ (2018, 8, emphasis added). Yet, the research base is far more definitive than acknowledged in the report, with recent papers arguing that air pollution is the second most significant cause of NCD-related mortality after tobacco (Neira et al., 2018, Prüss-Ustün et al., 2019). The need to tackle the environmental determinants of NCDs are, these authors argue, essential as effective action also holds the potential to mitigate climate change, reduce health inequities and produce economic co-benefits. But in *Time to Deliver*, the environment is reduced to a target more concerned with the landscape of regulation than ecological quality:

Governments must take the lead in creating *health-protecting environments* through robust laws, where and when necessary, and through dialogue, where appropriate, based on the “health is the priority” principle, including clear objectives, transparency, and agreed targets (WHO, 2018c, 23, emphasis added)

The report’s underplaying of the environmental drivers of NCDs is more curious given the backdrop of significant global health activity on the topic. For example, in May 2015, the WHO adopted resolution WHA68.8 on ‘Health and the environment: addressing the health impact of air pollution’ and a ‘draft road map’ for the global response to the ‘adverse effects’ of air pollution was adopted at the 69th World Health Assembly in 2016. Air pollution appears in a number of SDG targets across the health and urban spheres, with the WHO keen to ‘leverage the health sector to raise awareness of the health benefits of air pollution reduction measures’ (WHO, 2019). This aspiration is brought out even more clearly in the draft road map’s ‘theory of change’, which sets out the need for the health community to reframe ‘action on air pollution as a public health issue’ and to use ‘supplemental arguments of health benefits to further promote shared agendas’ (WHO, 2016, 12). The need to yoke together the pollution, climate change, development and global health agendas was also made clear by the report of the *Rockefeller Foundation-Lancet Commission on Planetary Health* (Whitmee et al., 2015), the *Lancet Commission on Pollution and Health* (Landrigan et al., 2018, Das and Horton, 2018)

and the UN Economic and Social Council's recommendation that pollution be an express target for the UN's interagency Task Force on NCDs (Fuller et al., 2018). Given this research and policy backdrop, *Time to Deliver's* omission of environmental risk is even more notable.

In 2017, WHO released its report on *Preventing Noncommunicable Diseases by Reducing Environmental Risk Factors* in which it argued that 23% (or 12.6 million) of all global deaths are 'linked to the environment' (2017, 2) and of these, two thirds are caused by NCDs (in particular ischaemic heart disease and stroke). These statistics are rendered more urgent because the majority of NCD deaths attributable to environmental risk factors are in middle-income countries where health risks are further worsened by the lack of effective standards, regulation and enforcement (Tousignant, 2018). The rampant rise in chemical production and use and the global export of waste to a growing multitude of southern 'sacrifice zones' (Lerner and Brown, 2010) renders toxic exposure both inescapable and thoroughly unquantified. However, just three months after the disappointments of *Time to Deliver*, the preliminary materials and agenda for the UN HLM listed air pollution as a component of newly (and quietly) christened 5 x 5 (Linou et al, 2018). With this comes a marked shift in the nature of NCDs as a "problem", their aetiological narratives and causal pathways (Kelly and Russo, 2018), the spectrum of commercial and public interests that have a stake and responsibility for addressing them, and, as a result, how we think about NCD prevention. This recalibration has not yet been explored within the health, environmental or social scientific literature. With 'behaviour' no longer the problematic that has long united the NCD discourse (Kelly and Russo, 2018), 'a revised paradigm is required for evaluating and prioritising the environmental contribution to human illness and the associated costs' (Grandjean and Bellanger, 2017, 7). Here I want to consider whether the conceptual scaffold of toxicity and the refreshed optics that it offers might forge a helpful path to this goal.

Toxicity and the new optics of NCDs

i. The language of toxicity

To be “toxic” is to be poisonous, bad, unpleasant, unacceptable or harmful. It also denotes something that causes harm *over a long period*. Its metaphorical ambidexterity, rhetorical power and political utility (Buell, 1998) no doubt contributed to the term being Oxford Dictionary’s 2018 word of the year. In technical terms, toxicity denotes the *level* of poison in a drug or chemical and the *ability* of the substance to poison the body. It is therefore the *quality* of being poisonous and the *degree* to which a substance is poisonous. Toxicity thus invokes questions of risk, harm, thresholds, exposure, temporality and spatiality, but struggles with ascribing definitive, linear causality (Mah, 2017). As we now inhabit an era of ‘mysterious diseases and conditions and unclear aetiologies: lupus and other autoimmune diseases, a growing number of allergies, unexplained increases in autism and neurological and developmental disorders’ (Brown, 2007, 230), researchers and the public have been increasingly looking to environmental explanations for these debilitating unknowns (Langston, 2010; Lappé, 2016).

When we think of toxins, we most often think of poisonous chemicals (Waters, 2019), pesticides, herbicides or waste that can cause rapid death or long-term sickness (Singer, 2011, Benko, 2019). Yet, the inclusion of air pollution as a NCD risk factor draws attention to the health effects of ambient and inescapable air, the toxicity of which exhibits significant and complex spatial variations (Li, 2019). Moreover and as with all toxins, measuring individual exposure is inherently complex (Calvillo, 2018) and the process of ascribing health effects to air pollution is subject to significant uncertainty (Jerrett et al, 2018; Kwan 2018). In the broader environmental sphere, toxicity also draws attention to the effects of low-level, ubiquitous and lifelong exposures to insufficiently regulated domestic chemicals, plastics, cleaning and gardening products, cosmetics, building materials, electronics or contaminated drinking or

ground water (Cordner, 2016). Air pollution is thus an effective entry point for a reinvigorated and long-overdue concern with an array of health-environment interactions in the context of NCDs (Nash, 2006).

For example, research on carcinogenesis has long grappled with the role of the environment versus that of lifestyle (Davis, 2007, Mukherjee, 2010), especially given rising incidence of cancer rates across the globe and the inability of lifestyle-related risk factors to fully account for this shift. Indeed, researchers have argued that roughly 40% of cancer cases can be attributed to environmental risk factors (Clapp et al., 2008, see also Jain, 2013) and, crucially, that such ‘involuntary exposure’ to environmental carcinogens (including radiation, xenochemicals and micro-organisms) may be systematically under-estimated (Irigaray et al., 2007). This is further reinforced because ‘while lifestyle-related factors are usually well determined and thus accessible to epidemiological studies, cancer-causing agents, because they are multiple, diverse and diffuse in the environment, are more difficult to identify and recognize and therefore evidence through classical epidemiological methods’ (*Ibid*, 641).

Exposomic concern with an individual’s lifetime toxic exposures (through their internal biomarkers and external stressors) and their relationship to health (Wild, 2005, Prior et al., 2019) or biosocial ideas of health (how social, cultural, economic and biological factors interact throughout the life course to produce differential health outcomes) (Singer et al., 2017, Singer and Clair, 2003) demonstrate that there is a chronicity and spatiality to the complex causal web that produces morbidity and that despite rapid advances in the models and technologies of exposure measurement, significant unknowns remain (Nah, 2017; Kwan 2018). By extension, this means that causality remains inherently uncertain as definitive proof of the multiple pathways and mechanisms of the pathogenesis of NCDs are almost impossible to attain (Frickel and Vincent, 2007). This is not least because toxins, as Fortun (2011) argues, evade linear models of causality and are instead ‘looped and entangled phenomena’ (Roberts, 2017, 615).

For this reason, as the NCD category widens from behaviour to the risks of environmental exposure, the frame of toxicity not only draws attention to the broad array of environmental exposures that might result in illness, but also the significant barriers to absolute certainty with respect to causality (Cordner, 2016, Boudia and Jas, 2014) that contrasts sharply with the relative certainty that characterises lifestyle-driven aetiological narratives.

The science of toxicology examines the nature, detection and effects of poisons, as well as the measurement and analysis of substances classified as toxins in the body (i.e. xenobiotics). In the context of NCDs, environmental toxicants might include: tobacco smoke, traffic-related pollutants, polycyclic aromatic hydrocarbons (from coal, tar or biomass burning), endocrine-disruptors, heavy metals and bioaerosols (Sly et al., 2016, Senanayake and King, 2017). Given that toxicologists generally study the effect of one chemical on the body in isolation, the field struggles to analyse the interactions between the multiple toxicants that compose everyday exposures and health outcomes (Tousignant, 2018). Toxicologists study dose-response relationships, with the assumption of increasing severity of effect as the dose increases, with the effect determined by the intrinsic toxicity of a substance, exposure conditions and the response of the host (Borowy, 2016). In toxicological lexicon, the ‘threshold dose’ is the dose level above which an adverse effect is encountered, with the assumption that the higher the dose, the more severe the response, generally presented as a sigmoid curve. The precise dose-response curve for a certain toxin thus allows toxicologists to predict a somatic response at a given level of exposure. As with many of the central tenets of toxicology, these have been formulated from animal or cell studies using high doses and then extrapolated back to hypothesise the effects of lower doses on humans, with varying degrees of accuracy and success.

However, and as Fortun (2011) suggests, toxicology’s ‘established paradigm’ has reached its methodological and conceptual limits. One reason is that toxicology has little way to account

for what Nash calls the ‘radical contingency of exposure’ (2006, 188) in which health effects and disparities are determined by a complex variety of factors, that have both spatial and temporal dynamics (Kwan 2018). For example, low doses during key ‘windows of susceptibility in foetal development can have far greater effects than higher doses in adults’ (Sly et al., 2016, 7). The threshold marks the point at which the body’s ability to detoxify a xenobiotic or repair a toxic injury is exceeded and, therefore, the point at which a toxin becomes a ‘safety concern’ that needs to be regulated. The question of human safety at sustained exposure levels that fall below such threshold doses is one of the main concerns raised by the environmental health and justice movements and, with the inclusion of air pollution as a risk factor for NCDs, must now be an even broader political and regulatory concern. Yet, and as Fortun (2011, 5) makes clear, ‘industrial culture [cannot] deal with [the kind of] intersectionality’ that characterises the variegated and inequitable topography of contemporary toxic exposures.

When it comes to the environmental risk factors for NCDs, exposure is generally sub-threshold and sub-clinical but may cause cumulative damage such that, at some point, the damage to the body and its cells becomes toxic and manifests as chronic illness. This exposure can start *in utero* (or even pre-conception) causing developmental toxicity, mutagenesis and later carcinogenesis (see Lappé, 2016). When bodies are exposed to more than one toxin, the effects can be additive, synergistic or antagonistic, but predicting these can be incredibly hard especially given differences in the ways that different people react to and process toxins. Such ‘toxic layering’ (Goldstein and Hall, 2015) fundamentally complicates the ascription of causality, especially as environmental toxicology and epidemiology struggle to define patterns of influence once toxins start to interact across space and through time (Fortun, 2011). For NCDs, toxic layering introduces ‘multiple and recursive possibilities’ (*ibid*, 651) in which environments are far more than settings within which people make lifestyle and behavioural

choices. Instead, thinking about NCDs through toxicity and its complex ecologies helps demonstrate – in painful and frustrating ways – just why rates of NCDs continue to rise and why prevention measures around behaviour change have proven so ineffectual for so long. Chronic illness is not a failure of individual will, but rather we are sickened by our collective enshrouding in emergent toxic possibility. Just as Ulrich Beck has argued, ‘reduced to a formula: poverty is hierarchic, smog is democratic’ (1992, 32). While people are clearly affected by pollution in differential and unjust ways; low-level chronic exposure to environmental toxins occurs across the social gradient through the layering of ‘invisible harms’ (Goldstein and Hall, 2015). The ability of individuals to reduce their exposure to the invisible is further limited by the ‘inherent uncertainty’ of toxicity and the resultant ‘risk frames’ that individuals and communities use to anchor the approaches they take to protecting themselves against environmental hazards (Auyero and Swistun, 2008).

Until recently, a fundamental tenet of toxicology has been that exposure below threshold levels is safe with the result that the kinds of long-term chronic exposures explored here have been underexplored and remain little understood to either toxicologists or epidemiologists (Davis, 2002). In such cases, evidentiary or ‘toxic uncertainty’ (Auyero and Swistun, 2008) emerges when multiple mechanisms underlie individual or population harms and there is a disjuncture between lay, expert and political readings of the relationship between risks and harms. More broadly, ‘if society is indeed entering an age of toxic layering in which multiple mechanisms underlie environmental harm, parsing causality becomes a daunting project’ (Goldstein and Hall, 2015, 652). Moreover, ‘the multiplicity of toxins in the environment, when coupled with the complexity of human habits (alcohol and tobacco use, for example), makes it deeply challenging for scientists to establish through peer-reviewed empirical research that any one substance has caused a particular harm’ (Goldstein, 2017, 322). Beck also raises this problematic of ascribed definitive causation in *Risk Society*, in which he argues that ‘it is

obviously impossible to bring individual substances into direct, causal connection with definite illnesses, which may also be caused or advanced by other factors as well...Anyone who insists on strict causality denies the reality of connections that exist nonetheless' (1992, 63). The inherent uncertainties of this research realm and the strategic unknowns and ignorance that can result have been the topic of a suite of fascinating sociological engagements (Frickel and Vincent, 2013, Roberts et al., 2008, Frickel and Vincent, 2007, McGoey, 2012). This is further exacerbated by a 'lavishly funded army' of commercial 'doubt producers and doubt disseminators' (2011, 40; see also Oreskes and Conway, 2010) who twist uncertainty and complexity into arguments for regulatory inaction, delay and infinite calls for further research. With this comes important political questions of where responsibility for ameliorating the effects of our toxic everyday really lies.

ii. (re)Locating responsibility

Public health is under siege and the public's health is under threat from the 'commercial determinants of health' (Kickbusch et al., 2016). Stung by the experience of Big Tobacco, the public health community is on high alert, mobilising the language of war and issuing a 'call to arms' (Demaio and Marshall, 2018) to undertake 'assertive advocacy' to resist and counter the tactics used by industries selling 'harmful commodities' and protect the public's health (Yach and Bettcher, 1998). The language of a recent paper is indicative of the fight: 'If you are working to improve public health and the environment, you need to know what your *opponents* are up to' (Moodie, 2017, emphasis added). In their staunch opposition to the many of the products and practices of industry, the public and environmental health movements share a long history of anti-corporate activism, even if the methods and targets of their action have been very different. Rachel Carson's *Silent Spring* (1962), for example, brought to light the

threat posed by pesticides and the role of the chemicals industry in manipulating science, evidence and policy and in resisting regulation. While most commonly associated with the environmental justice and conservation movements, *Silent Spring* was also a profoundly important public health intervention, characterising the chemical industries and their products as pathogenic, but also untrustworthy, unethical, and motivated by profit over public good (Buell, 1998). As we reach a powerful inflection point in two interlocked “crises” - climate change and the global rise of noncommunicable diseases – public health concern with ‘industrial epidemics’ or ‘corporation-induced diseases’ (Jahiel, 2008, Jahiel and Babor, 2007) where the ‘vectors of spread are not biologic agents, but transnational corporations’ (Moodie et al., 2013, 671) has widened and hardened. In many respects, opening the NCD category to the environmental determinants of health has merely added extra culprits to a hitlist of nefarious industries that has, until now, coalesced around food, tobacco and alcohol but would now have to include car manufacturers, the chemical industries, commercial farming, cosmetics companies and many more.

This difference is most marked for the fact that the lifestyle frame that united the four *shared* and *modifiable behavioural* risk factors for NCDs has been splintered by the addition of air pollution. And, as Kelly and Russo argue, ‘disease is conceptualised as the consequence of exposure to a pathogen or other preceding noxious agent... The principle is that there are causes and, more specifically, the preceding *noxious causal agent* in the case of non-communicable diseases is human behaviour’ (2018, 83). This, in turn, has also provided the legitimisation frame through which the food, alcohol and tobacco industries have sanctioned their products as socially accepted and tolerated risks that are ‘safe’ when consumed moderately within a broader landscape of ‘balanced lifestyles’. Just as Beck argues, ‘the appeal to “responsibility” is the cynicism with which the institutions whitewash their own failure’ (2006, 336). However, when consumption is replaced by a toxicity-driven paradigm of *exposure* (in the context of air

pollution), then the trope of individual responsibility that has served policy makers and business so well is undermined. Even if, so far, advice to prevent health harms from air pollution is for ‘high risk’ individuals to ‘minimise’ or ‘avoid’ exposure to hazardous pollution events, this invokes different questions of risk and agency than those of consumption.

The disciplinary siloes of public health, where specialists in alcohol or tobacco or nutrition may not necessarily think through the broader ecologies within which these products are manufactured and consumed has also reinforced the tendency to link NCD risk factors to a clearly defined set of ‘commercial determinants of health’ (Kickbusch et al., 2016, McKee and Stuckler, 2018). For example, while those in public health explore ‘product-based NCD risk’ (Lencucha and Thow, 2019) from alcohol, tobacco and food products and the ‘product environments’ (*Ibid*) that encourage their consumption (i.e. marketing, formulation etc), the actual physical environment (in an ecological or contextual sense) is less frequently subjected to critical reflection (Herrick, 2019). This approach reflects an anti-corporate paradigm that is closely tied to disease causation models that contend that, ‘health outcomes are determined by the influence of corporate activities on the social environment in which people live and work: namely the availability, cultural desirability, and prices of unhealthy products. The environment shapes the so-called lifeworlds, lifestyles, and choices of individual consumers—ultimately determining health outcomes’ (Kickbusch et al., 2016).

In this formulation, the ‘environment’ is the context within which corporate activities and political economic systems conspire to mould the consumption of inherently dangerous products. And, despite the call by some public health advocates for a return to ‘the classic public health approach of environmental intervention that was so effective in achieving marked health improvements in the 19th and early 20th centuries’ (Jahiel and Babor, 2007), here too the ‘environment’ refers to political economic rather than ecological risks. The same authors may want to shift attention to ‘the “upstream” sources of the damage, as opposed to attributing

[for example] alcohol-related problems exclusively to the personal behaviour of the individual drinker’, but the ‘upstream’ still refers to issues of marketing, advertising and product development rather than, for example, the widespread ecological impacts of alcohol production (i.e. water contamination, plastic packaging, inadequate waste disposal) and the health effects of this. Indeed, the public health vision of the causal linkage between the unfettered rise of these industries, their products and activities and NCDs is made clear by Hastings:

Tobacco has remained such an intractable problem only because our economic system allows free ranging corporations to market it. The same applies to the other two “industrial epidemics” that constitute such a large share of the public health burden: alcohol misuse and obesity. In each case, evocative promotion, ubiquitous distribution, perpetual new product development, and seductive pricing strategies are used to encourage unhealthy consumption... The consequence has been the inevitable escalation of lifestyle illnesses such as cancer, heart disease, cirrhosis, and diabetes’ (Hastings, 2012, e5124).

Here, ‘lifestyle diseases’ – a misleading synonym for NCDs – are the result of the *political economic environments* that enable companies to produce and freely sell their products to consumers. Yet, the risk of those same ‘lifestyle diseases’ - cancers, heart disease and diabetes - can also be increased by *exposure* to ambient air pollution (Campbell-Lendrum and Prüss-Ustün, 2019), a relationship that is not mediated by ‘irresponsible’ consumption, but rather the inescapable, invisible and often-unmodifiable exposures of everyday life. The predominant aetiological frame of NCDs as lifestyle and consumption-driven also hints at why other ‘noxious’, health-harming industries have been less frequently explored as problematic within the NCD category. The manufacturing, chemicals and waste industries, for example, have long been the target of outspoken opposition by environmental health campaigners. Activist-author Steve Lerner and sociologist Phil Brown, for example, have explored the manifold ‘sacrifice zones’ of toxic pollution across the US and the development of the environmental justice movement from its origins in efforts to prosecute those responsible for the health and environmental effects of toxic contamination from a PCB landfill in Warren County, NC in the 1980s (2010). Anthropologist Merrill Singer (2011) and more recently geographer Thom Davies (2018, 2019) have examined the nature of exposure, science, politics and community

activism of Louisiana's infamous 'Cancer Alley', a region of the US where toxic exposure is punitively stratified along race and class lines. In a fascinating counterpoint to the US-centric literature, Alice Mah and Xinhing Wang (2019) have explored similarly stratified exposure to petrochemical pollution in Nanjing, China. This research domain is united by an interest in and support of 'citizen science' and 'popular epidemiology' (Richter et al, 2018) as exposed residents try (with varying and limited degrees of success) to draw on the same 'molecular bureaucracy' that fails to protect their health (Hepler-Smith, 2019) to seek justice from corporate polluters. In stark contrast, public health advocates concerned with the behavioural risk factors for NCDs have rarely targeted the toxics industries or been particularly animated by ideas of popular activism or advocacy. And yet these constituencies are largely united in their concerns over the *same* diseases. While these divergences may have much to do with the longstanding disciplinary separation between public and environmental health (Berridge and Gorsky, 2012), it is also clearly an outcome of markedly different aetiological paradigms between the two disciplines – consumption versus exposure – and, therefore, differing conceptualisations of risk, hazard and responsibility.

For industry itself, the aetiological paradigms of consumption and exposure have proven to be incredibly useful in eschewing and shifting blame (Petticrew et al., 2017). In effect, industry has become adept at paradigm hopping knowing that proving that a specific industrial chemical led to a specific illness is notoriously difficult and even harder to prove in court, given the burden of proof of harms rests on the afflicted rather than industry (Lerner and Brown, 2010; Richter et al, 2018). Indeed, industry has long mobilised the inherent uncertainty of human-environment interactions and their effect on health to their strategic advantage in deflecting calls for greater regulation. Similarly, the 'unhealthy commodities industries' have pointed to the inherent complexity of lifestyles and the multiple environmental contexts inhabited by individuals and the difficulties in proving the causal influence of their products alone in

producing disease. Here, the aetiological paradigms of consumption and exposure are used to deflect blame from industry and back onto individuals. However, with *ambient* environmental exposure (rather than the acute toxic ‘point exposure’ often associated with environmental justice cases against industry) and lifestyle now rolled into the NCD category, industry’s capacity to paradigm hop may start to be constrained. However, as the NCD field necessarily shifts to bring together diverse research and advocacy traditions (Linou et al, 2018) and a new host of commercial foes within an international political context where “partnership” with industry is the order of the day (see *Time to Deliver*), delivering NCD prevention looks set to become even more fraught (see Rosner, 2009).

iii. The new optics of NCDs

When the term ‘optics’ is used in global health, it usually denotes the study of light and human vision. Yet, in the murky worlds of politics and business, optics has quickly risen to lexical primacy. Worrying about the ‘optics’ of a situation, decision or policy – or how it *appears to* or is *perceived by* people when filtered through the media and, crucially, the political consequences of this - is an inescapable political pastime and now a realm of specialist consultancy. That concern with the ‘optics’ is often greater than the substance of what actually happened says much about our contemporary, visual, media-saturated life (Nixon, 2011). It also says much about the persistent disconnect between those in and with power and the public. Deriving from the French *optique*, which means both the science of optics or a perspective or point of view (Zimmer, 2010), the term scientises public perception and attempts to make it amenable to prediction. While generally applied to political escapades and events, the term might equally be used to refer to other phenomena for which perception and appearance are as significant as ‘reality’. Optics, moreover, also draw attention to the difficulty in aligning public

perception with political intention. Or, in the case of health, aligning epidemiological risk with public understanding and, as a consequence, the necessary behaviour.

Here, rather than draw on the far more frequently used concept of framing (Entman, 2007), I want to explore how the idea of ‘optics’ might be usefully applied to the category of NCDs. The distinction between the two is worth drawing out: framing refers to instrumental and intentional efforts to cast an issue or a problem in a particular way in order to elicit a particular response. Optics, by contrast, draws attention to how something appears or is received, and while this may be unpredictable or unanticipated, public perception can be moulded. Moreover, while framing is a largely linguistic pursuit, optics also draws in the visual, the visceral and the affective which is now arguably of far greater significance in our image-saturated communications. I am also drawn to this nomenclature because amid the UN High Level Meetings and the multitudinous journal papers, commentaries and editorials decrying the lack of concrete progress on addressing NCDs (Blundell and Hine, 2018, Horton, 2017, Horton and Sargent, 2018); the term ‘optics’ is never used. This omission is important because as Thom Davies, drawing on the work of Rob Nixon, has argued, ‘situations of slow peril – such as pollution, climate change, or deforestation – have their own situated, contested and problematic optics that deserve attention’ (2019, 11). The ‘malignant neglect’ (Stuckler and Basu, 2013) of NCDs can arguably be traced to how the category is understood, perceived and ‘seen’ by the public as well as the vastly different communities of practice that need to work intersectorally to address the issue (Allen, 2017, Allen and Feigl, 2017a, 2017b). Given this profound nosological challenge, what optics emerge when NCDs are viewed through the lens of toxicity? In order to explore this, I turn to recent work in public health arguing for greater recognition of the toxic properties of alcohol and food.

Among the risk factors for NCDs, a group of public health researchers have called for alcohol to be considered ‘toxic’ and regulated as such due to the acute effects of intoxication and organ

toxicity (Babor et al., 2003; Kypri and McCambridge, 2018). They argue that alcohol should be regulated in the same way as other harmful drugs and psychoactive substances to reflect and communicate the fact that it is effectively a ‘dangerous poison’ with acute and chronic effects. Here, toxicity is used as rhetorical hook to change public perceptions about relative risk and to challenge the narratives of a right to pleasure long associated with drinking. It is also used to spur greater levels of regulation and, perhaps more importantly, ensure that the purveyors of these toxins are subjected to far greater regulation than is currently the case. Public health advocates suggest that alcohol is part of broader set of industrial epidemics in which the ‘policy focus’ should be shifted from ‘the agent (i.e. alcohol) or the host (e.g. the problem drinker) to the disease vector (i.e. the alcohol industry and its associates)’ (Jahiel and Babor, 2007). However, while they agree on this, they often come unstuck on the question of threshold levels of ‘safe’ exposure/ consumption, itself a core component of the science of toxicology. To be toxic is only technically such above a specific dose, a question that has long riled and divided the public health community on the topic of alcohol (Lovatt et al., 2015), with government guidelines treading a delicate line between the science of harms and the public’s perceptions of relative risk. Yet, recent research has highlighted the broader environmental harms perpetuated by the alcohol industry - as major consumers of limited resources such as water, significant contributors to greenhouse gas emissions, use of environmentally unsustainable crops and the creation of toxic by-products that can have profound ecosystem effects. Alcohol understood in terms of environmental harms and toxic exposure (often borne disproportionately by countries in the global south) produces a new form of optics, of social (in)justice, (in)equity, resource extraction, industrial rights to water use in contexts of drought and exposure as a driver of health harms. As such, it disrupts the optical paradigm of individual irresponsibility in favour of a broader, ecological view of the diverse socio-spatial instantiations of alcohol harms.

Toxicity has also been associated with foods, most commonly in relation to food safety scandals. However, in the context of NCDs, research and advocacy have also elided the category of “ultra-processed foods” (UPF) with notions of toxicity (Monteiro, 2011). Defined as ‘industrial formulations made mostly or entirely from substances derived from foods and additives, with little, if any, intact food’ (Rico-Campà et al., 2019), UPFs have long been a cause of public health concern, but most commonly associated with a heightened risk of weight gain in the public imagination (Canella et al., 2014, Juul and Hemmingsson, 2015, Mendonça et al., 2016). However, research on UPF now shows that increased consumption is associated with an increased hazard of all-cause mortality (Rico-Campà et al., 2019). Two recent French cohort studies also found an increased risk of breast and bowel cancers as well as the symptoms of Irritable Bowel Syndrome among those consuming the highest amounts of UPF (Fiolet et al., 2018, Schnabel et al., 2018). Further studies have found explicit links with cardiovascular disease (Srouf et al., 2019).

While being mindful of the caveats associated with any study of this kind around the clear ascription of causality, the fact of even considering the prospective relationships between UPFs and cancers (rather than simply body weight) echoes calls for more ‘ecological nutrition’ (Mason and Lang, 2017). Echoing alcohol, it also reflects the fact that now, ‘*how* food is made may be more important to dietary health than previously thought’ (Guthman et al., 2014, 46, emphasis added). No longer is the nutrient profile or the calorie content of food the sole target, but the ecological contexts within which food is produced and the environmental and health externalities resulting from industrial food production, packaging, transportation and chemical processing. The matter is no less pressing for the fact that as noted in a recent editorial in the *BMJ*,

We are a long way from understanding the full implications of food processing for health and wellbeing. Care should be taken to transmit the strengths and limitations of this latest analysis to

the general public and to increase the public's understanding of the complexity associated with nutritional research in free living populations (Monge and Lajous, 2018)

In all this research, one of the key unknowns is the biological pathways by which, for example, UPFs influence health outcomes. Theories include their effect on the gut microbiome and their ability to feed “bad” bacteria and alter the delicate equilibrium (type and variety) of the gut flora (Zinöcker and Lindseth, 2018). Some have asserted possible pathways through their high refined carbohydrate content or lack of fibre which might result in chronic inflammation, a reduced ability to fight off infection and, in turn, higher rates of some chronic diseases. The emulsifiers used in many UPFs have also been shown to illicit a chronic inflammatory response in animals (Chassaing et al., 2015). As nutritional scientist Carlos Monteiro has noted, some processes used to produce UPFs may then be toxic to humans, by being ‘either carcinogenic, or harmful and addictive, or else identified as intensely pathogenic’ (2011, 499). These processes thus require close regulation. He goes further to argue that the process of hydrogenation – by which hard fats such as margarine or the now-vilified trans fats are produced – are so toxic that they should be banned altogether, as should some ‘cosmetic additives’ (see also Thornton, 2018, Ganguly and Pierce, 2015). In a commentary piece, Monteiro further argues that the term ‘toxic’ should rightfully be applied to ‘processes used to make ingredients or foods that directly or indirectly are carcinogenic, or addictive, or else acutely pathogenic, because of the process itself’ (2011, 503). These are new optics – no longer is food demonised on the basis that it might increase bodyweight and, as a result of this, impair health. Rather, its very creation, the chemicals contained in its packaging and its transit to market, furnishes it with properties that are inherently damaging to human health and natural environments. Crucially, these are issues that, in the context of increasing environmental concern and climate awareness, resonate with consumers in potentially more powerful ways than moralised exhortations to ‘eat healthily’.

An optics of toxicity opens new questions about rights and responsibilities in relation to NCDs. In essence, toxicity makes it clear that the agents of change must be the *sources* of contaminants. It also provides a window of opportunity to bring a well-established – and growing – environmental justice movement to bear on issues (and companies) that have courted a more ambiguous public response. The depth of public health opposition to big food and alcohol is not shared to the same extent by a public caught between desire and awareness of the health implications of their products. This tension is an important entry point into a new optics: some individuals can “optimise” their behavioural and lifestyle choices, but no-one can stop the by-products of industrial food production leaching into local ecologies. There is a very real limit to which anyone can control their exposure to toxins they cannot see, know or understand. The risk of such ‘toxic trespass’ is even more so among the poor, the young, the marginalised and those living in the most environmentally degraded areas, as environmental justice campaigners have long argued (Lerner, 2005, 2010, Shevory, 2007). That the causes of fatal or debilitating maladies cannot be definitively traced is cause for concern not just for what it says about the limits to biomedicine and epidemiology, but also because it reveals ‘that the ways we die and the diseases that afflict us are, in large measure, reflections of the world we build and the environments we create’ (Markowitz and Rosner, 2013, 272). As a result, the intuition and feeling that chronic illnesses emerge from risks far outside individual control and in ways that people cannot predict demands that we switch the optics from consumption to exposure. Doing so reflects responsibility back onto producers rather than consumers, shifts the focus and blame from demand to supply and, in an ideal world, would produce a regulatory realm in which the purveyors of toxins must prove the pre-release safety of their products rather than asking those suffering the effects of toxic exposure to prove the definitive source of harms. Doing so brings the objects and targets of both the environmental and public health movements

into the same political and discursive orbit, an essential step if any advance is to be made in tackling NCDs.

Conclusion

This paper has explored the recent addition of air pollution as an environmental risk factor for NCDs. In so doing, I have argued that we have reached a crucial inflection point where the optics of NCDs need to shift from individual consumption to toxic exposures. The lack of effective political action or accountability on NCDs (Stuckler and Basu, 2013) and the concomitant, continued rise in global prevalence suggests the total failure of the optics of lifestyle and behavioural risk. As Rachel Carson so aptly noted in *Silent Spring*, ‘it is human nature to shrug off what may seem to us a vague threat of future disaster’ (1962, 169), and the NCD agenda has struggled against the anomie of long-term, invisible and largely incalculable threat. Thinking about NCDs *through* and *as* toxicity may hold the potential to shift this. As Liboirin (2018, 341) argues, ‘while toxicity is embedded in multifarious relations of power, it also has the potential to invent alternative political relations: The precariousness of toxic worlds enables the formation of resistances, coalitions and practices that expand the inventory of what politics means and does in late industrialism’. Public health engagement with NCDs has, thus far lacked the concern with social (in)justice, activism and advocacy that has long characterised the environmental justice movement. Making toxicity ‘knowable and accountable’ through the policy sphere of NCDs might go some way to opening-up much-needed spaces for the kinds of ‘citizen intervention’ (Cavillo, 2018) that could ensure greater global action.

The lifestyle frame has enabled the commercial determinants of health to eschew responsibility and evade regulation through the invocations of the mantra of individual freedom, choice, and balance (Herrick, 2009). The controversy over *Time to Deliver*’s failure to include an express recommendation on states adopting sugar-sweetened beverage taxes to reduce consumption is

a case in point here. Arguing for the need for a clear evidence base (while contesting the substantial, existing evidence base) and for a policy of consumer education; the episode demonstrates the strategic power of uncertainty and complexity (Petticrew et al., 2017) as well as the use of education as a solution to the broader problem of lifestyle. Toxicity and the addition of air pollution to the NCD mix fundamentally complicates this as individuals are now *passively exposed*, rather than *active consumers* whose freedoms and agency must be protected. This means that new types of freedoms must be protected – rather than *freedom to* consume and take (informed) risks, individual and community *freedom from* harmful exposure must be ensured. The locus of responsibility for this is squarely with producers and regulatory bodies. Within the lifestyle frame offered up by the NCD paradigm, it is tempting to somehow consider that diseases such as breast cancer are fundamentally different when produced by chemical exposure than when by alcohol consumption (Brown, 2007, Davis, 2007). But consumption is far more easily enumerated than the multiplicity and ephemerality of exposure. Methodologically, it is simpler to survey food intake, drinks consumed, cigarettes smoked (and then account for reporting bias) than it is to quantify the temporal and spatial dynamics of multiple chemical exposures across the life course. Toxicity allows for (and expects) uncertainty, while behavioural explanations treat uncertainty as evidence of individual failure, guilt or complicity. Exposure, therefore, reworks an optics in which moral judgment is more firmly attuned to causes rather than their somatic effects.

The problem of NCDs is arguably most acute in countries of the global south where most global chemical production will soon be concentrated, where environmental degradation is most acute and where restrictions and regulations to safeguard human health and the environment are most lacking. Often health regulations are by-passed as they are seen as being in tension with economic growth, jobs, prosperity and development (Grandjean and Bellanger, 2017). This is also true in many countries of the global north, especially the US which has long been at the

vanguard of both public concern over toxins and an attendant failure to adequately regulate their use (Davis, 2002). Across the global south, accurate data on exposure and harms remain sparse, but arguably of paramount importance if the current gaps in the GBD are to be sufficiently addressed to enable the full extent of environmental risk factors to be quantified, understood and acted on. The changing composition of the NCD category makes us ask important questions of how and why the environment and health got so disentangled in earlier narrations of NCDs (Nash, 2006), especially given that the environment is both essential to good health and a threat to it. The recent categorical shift set out in this paper is thus an invitation to re-integrate the two and, in the process, offers up the possibility of a powerful new optics of toxic health risk that frames uncertainty as a reason for action, rather than *inaction*. The category of NCDs has long been critiqued for lacking salience and meaning to those outside expert public and global health circles (Herrick, 2019) and it is doubtful that the inclusion of air pollution renders NCDs a more legible category across increasingly diverse communities of interest (Bowker and Star, 2000). However, in making it clear that risk cannot be simply parsed by reference to individual behaviour and should instead be located in the miasma of environmental risk; the concomitant call for responsibility and restraint in consumption as a solution to the conditions that ail us is shown for what it is: a destructive act of mass distraction.

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